# PERFORATION OF THE INTERVENTRICULAR SEPTUM OF THE HEART

## WITH REPORT OF A CASE*

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Perforation of the interventricular septum of the heart, aside from cases of congenital malformation, in the great majority of cases is due either to rupture of an interventricular aneurysm or to ulcerative endocarditis. Perforation from other causes, such as rupture of a hydatid cyst,[1] has been described but such cases are relatively very rare.

### CARDIAC ANEURYSMS

Cardiac aneurysms have long been the subject of reports in the literature, and many excellent monographs have been written on the subject. It is said to have been first described by Guzman Galeati in 1757.[2] Also Thurman[3] reports a case from the manuscripts of John Hunter described in 1757. The first monograph was written by Breschet in 1827.[4] Pelvet,[5] in 1867, gave an excellent discussion of the subject. Bourland,[6] in 1904, without giving references, reports having collected 147 cases from the literature. Osler and McCrae[7] refer to 135 cases, and Friedlander and Isaacs[8] reported a case recently.

Aneurysms of the heart may form in the valves, walls or interventricular septum. They may be acute or chronic. Acute aneurysms are associated with acute endocarditis and will be considered under the discussion of that condition. Chronic aneurysms are, in the great majority of cases, the result of chronic myocarditis and fibrosis. As a rule, the chronic myocarditis is due to arteriosclerosis of the coronaries, and the anterior branch is the one most commonly affected. The left ventricular wall, near the apex, is the most usual site, as would logically follow as the result of the anterior coronary being most often affected. The aneurysmal formation may vary from the slightest aneurysmal

*From the Medical Service Massachusetts General Hospital.

1. Crowther, B.: Austral. M. J. **2**:362, 1880.
2. Cited by Pelvet.
3. Cited by Pelvet.
4. Cited by Pelvet.
5. Pelvet: Des Aneurysmes du Coeur, Paris. Delchage, 1867.
6. Bourland: Aneurysm of the Heart, Am. J. M. Sc. **128**:323, 1904.
7. Osler and McCrae: Modern Medicine, Ed. 2, Philadelphia, W. B. Saunders Company, **4**:482, 1915.
8. Friedlander, A, and Isaacs, R.: Interventricular Cardiac Aneursym with Heart Block, J. A. M. A. **75**:1778 (Dec. 25) 1920.

dilatation to a definite aneurysmal sac. The wall is thinned and the muscle tissue is largely replaced by fibrous tissue. There frequently are adhesions to the pericardium and rupture is fairly common.

Aneurysms of the interventricular septum differ in no essential respect from cardiac aneurysms in general, except as to location. According to Klein [9] they were not described until 1839. In contradistinction to cardiac aneurysms in general, they are most commonly found at the base of the heart, in the "undefended space" or pars membranacea. As the whole septum derives its blood supply from the anterior coronary artery which is most commonly involved in cardiac aneurysms, some additional reason has been sought for the occurrence of septal aneurysms at the base in the large majority of cases. Klein mentions gross abnormalities and increased pressure in the left ventricle, due to hyperthophy, acting on the weakest part of the wall. Others have discussed the character of the tissue forming this part of the septum which is fibrous in character. Also endocarditis is more frequent in this region due to easy extension from the valves, causing the formation of a weak spot and a subsequent aneurysm.

### PERFORATION OF THE INTERVENTRICULAR SEPTUM

Perforation of the interventricular septum due to ulcerative endocarditis has also been reported fairly frequently. Callender [10] reported a case in 1858, while Fournier,[11] in 1884, discussed the condition at length and reported a case of his own and seven others which he collected. Gennari,[12] in 1904, reported a case and gave a short bibliography.

For reasons which are similar to those given for the more frequent occurrence of septum aneurysms in the pars membranacea, perforation due to ulcerative endocarditis is also most likely to occur in that area. Proximity to the valves from which involvement often occurs by direct extension, the character of the tissue, the poor blood supply in that area, congenital defects, all make this area peculiarly susceptible to such an infection with resultant perforation.

The symptoms of perforation of the interventricular septum are indefinite and are not pathognomonic of the condition. In the cases of perforation of fair size, occurring acutely, there is the picture of sudden cardiac failure. When, as in the majority of cases, it occurs more

9. Klein, G.: Zur Aetiologie der Aneurysmen der pars membranacea septi Ventriculorum Cordis and deren Ruptur; Arch. f. path. Anat. **118**:57, 1889.

10. Callender: Med. Times & Gaz. Feb. 1858.

11. Fournier, H. C.: Etude sur les Perforations de la Cloison interventriculaire dans l'Endocardite Ulcereuse, Paris, 1884, Davy.

12. Gennari, C.: Sopra un Caso di perforazione del setto interventriculaire determinata de endocardite ulcerosa. Riv. Crit. di clin. med. **5**:717, 1904.

gradually, there is only a greater or smaller increase in the signs of cardiac distress which the patient already shows. The murmurs are not characteristic. Fournier speaks of cyanosis as an important sign but in Talamon's [13] case there was no cyanosis. Gennari says that the most important signs are pulsation to the right of the sternum, dilatation of the right ventricle, a murmur to the right of the sternum transmitted to the right and a thrill in the same area.

While cardiac aneurysms are fairly common, aneurysms of the interventricular septum are rather rare and perforation of the septum due to their rupture is even more rare. Including cases of perforation of the septum due to ulcerative endocarditis, a careful search of the literature since 1879 has yielded but twenty-two cases of perforation of the septum due to all causes, which were reported as such. Peacock [14] in 1854, reported a case in which the rupture also included the ventricular wall. Reinhardt, [15] in 1857, reported a case due to rupture of an aneurysm of the interventricular septum; Talamon cited a case in 1878; V. Buhl [16] has reported five cases and Klein and Lee [17] each one. Callender apparently reported the first case of perforation due to ulcerative endocarditis in 1858. Fournier, in 1884, reported one case of his own and seven others. Peron,[18] Fisher [19] and Gennari have each reported one case. In all of these cases the perforation occurred at the base of the septum and in none of them was there found a perforation similar to the one occurring in the case to be described below, in which case the perforation occurred near the tip of the septum a short distance above the apex.

## REPORT OF CASE

*History.*—R. B. (East Medical 238,660), a widowed, white woman, aged 52, entered the Massachusetts General Hospital Sept. 13, 1920. She was born in Pennsylvania and had lived in Massachusetts forty-seven years. Her family, marital and social histories were essentially negative. According to her past history, her general health had been good until about ten years previously.

13. Talamon, C.: Endocardite du Septum et Aneurysme interventriculaire; perforation de la Cloison; endocardite de l'infundibulum; Progrès méd. **7**:984, 1878.

14. Peacock: Tr. Path. Soc. Lond. **5**:102, 1854.

15. Reinhardt: Zur Anatome und Patholgie der dünnen Stelle in der Herzcheidewand; Arch. f. path. anat. **12**:129, 1857.

16. Buhl: Ztschr. f. Biol. 253, 1880.

17. Lee, A. E.: Case of ruptured aneurysm in the Ventricular Septum with Sudden Death; U. S. Naval Bull. **2**:49, 1908.

18. Peron, A.: Endocardite droit infectieuse post peurperil; perforation de la Cloison interventriculaire per une lesion unique ayant Amene une insufficiance tricuspidienne et insuffisiance aortique; Bull. Soc. Anat. de Par. **70**:198, 1895.

19. Fisher, H.: A case of Ulcerative Endocaritis involving the Pulmonary Valve, causing a perforation of the ventricular septum, gangrene of the nose and ears and multiple infarcts of the kidneys; Proc. Path. Soc. Phila. **6**:98, 1902.

Since then she had had frequent tonsillitis. She had entered this hospital on three previous occasions. The first time (East Surgical 204,937), in the fall of 1915, she complained of several attacks of epigastric pain, nausea, vomiting, headache, clay colored stools, dysuria, and frequency of urination. Operation showed only a slightly inflamed appendix which was removed. Her second entry a year later (East Surgical 210,450), was for a similar complaint plus backache and a vaginal discharge. At this time the urine showed sugar on two of five examinations and albumin three times. At her third entry, two years later (East Surgical 282,282), she complained of the same right upper quadrant pain plus moderate dyspnea on exertion and nocturia. Physical examination showed the heart not enlarged and slight sinus arrhythmia. The urine showed 2.0, 0, and 2.9 per cent. sugar, respecively, on three examinations. Following her discharge she was treated in the diabetic clinic of the outpatient department for about a year. She was placed on a restricted diet and did well, her urine on the last day she attended the clinic showing only the slightest possible trace of sugar.

On her present admission she stated that she had had slight dyspnea with mild anginoid pains, and slight palpitation when tired or excited, occasional orthopnea and edema for eight or ten years, and a dry, unproductive cough for years. She had been on a general diet since she had left the outpatient department eighteen months previously.

*Present Illness.*—The present illness occurred suddenly, ten days before admission, with substernal distress during moderate exertion, which had passed off in about twenty minutes. That evening, while in bed, she had a sudden, sharp, substernal pain, radiating to the back, shoulders and left arm. It was associated with dypsnea and orthopnea and lasted for twenty-four hours. It has recurred several times since then. For six days she has had increasing edema of the feet. Vomiting occurred three days before entrance, at first associated with the anginal pains. For three or four days her cough has grown worse but there has been no sputum.

*Physical Examination.*—On admission the following positive findings were noted: Obesity and slight cyanosis of the mucous menbranes. Lungs: Showed slight dulness at both bases posteriorly, with fine moist râles. Heart: Apex impulse was neither seen nor felt. The sounds were regular and rapid (140). There was a thrill at the apex and a blowing murmur over the whole precordium both of which seemed diastolic in time but it was impossible to determine the time accurately because of the rapid heart rate. The murmur was loudest to the left of the sternum, in the fourth and fifth interspaces and was transmitted to the axilla and the back. A very soft systolic murmur was heard at the apex. The pulses were equal, synchronous and of normal volume and tension. The arterial walls were not palpable and the brachials were not tortuous. The blood pressure was 125 systolic and 80 diastolic.

The leukocyte count was 14,000; hemoglobin, 80 per cent., and the smear and differential count were normal. The nonprotein nitrogen was 47.1 mg. per 100 c.c. of blood. The blood Wassermann was negative.

Treatment and Course.—The following day the urine showed 7.1 per cent. sugar. Carbohydrate was fed in the form of sugar water and she received an ampoule of digifolin intravenously. The note of that day says: "Cyanosis, pallor, blowing murmur at the fourth chondral space probably systolic, second sound not heard, rate rapid. Small amount of pleural fluid. History suggests coronary disease."

On the second day the carbon dioxid tension of the alveolar air was 17. The leukocyte count was 20.000. On the third day the apex rate was 180-190 with a high pulse deficit, apparently auricular fibrillation. The murmur over the precordium was definitely systolic and the sounds were faint. The electrocardiogram taken a short time after this examination showed simply a sino-auricular tachycardia, with a somewhat inverted T wave. One hour

later, the rate was 120 and perfectly regular. Paroxysms of aurcilar fibrillation were thought quite likely. The respirations were from 35 to 45 per minute. The digitalis was increased. The urine showed 2.17 per cent. sugar and a positive diacetic acid test. The blood sugar was 0.54 per cent. The following day her condition was much worse and she was practically unconscious. She was given orange juice and fat free milk and 400 c.c. of a 5 per cent. sodium bicarbonate solution intravenously. Acetone bodies in the blood were 3.72 mg. per 100 c.c. The patient grew rapidly worse and died that evening, the fourth day after admission.

*Necropsy.*—Necropsy 4,116, twelve and one half hours postmortem (abstract). The body is that of a white woman, 52 years of age, 162½ cm. long, well developed and stout. Head: Not examined. Trunk: Slight edema of the feet and ankles. Skin and Mucous Membranes: Pale. On section, subcutaneous fat in large amounts. Abdomen and contents essentially negative. Diaphragm: Negative. Pleurae: Right, a few adhesions to the pericardium and a few posteriorly. Left, a few adhesions posteriorly and one to the diaphragm. Trachea and Bronchi: Contain a moderate amount of reddish brown, frothy fluid. The mucoso is brownish red. Lungs: The tissue is slightly leathery, salmon colored and yields a considerable amount of thin, brownish red, frothy fluid. Pericardium: Negative. Heart: The heart weighs 310 gm.; a little enlarged. The myocardium, except in an area to be described, is of fairly good consistency, pale, brown red. The right ventricular wall is three to four mm. thick; the left ten. The columnae are fairly well marked on the right but flattened on the left. The right cavity is slightly dilated. The left ventricle, anterio-laterally to the right and opposite to the interventricular septum in its lower half, presents a pouch like dilatation 8x4½x2½ cm. The cavity on this side was otherwise negative. The auricular appendages are negative. Valve circumferences—mitral 10 cm., aortic 6 cm., tricuspid, 12 cm., pulmonic 8 cm. The aortic cusps show a slight amount of sclerosis. The valves are otherwise negative. Foramen ovale closed. Right coronary artery free, fairly capacious and shows only a slight amount of fibrous sclerosis. Left, first portion clear and shows only a very small amount of fibrous sclerosis but at a point about 3½ cm. from its origin the left descending branch is practically occluded by a fibrous plaque. The vessel and its branches beyond this point are very small and dwindle away in the wall of the dilatation previously mentioned. The myocardium in the region of the dilatation is about 3 mm. thick and shows a leathery fibrosis. At a point at the base of the dilatation, in the region of the interventricular septum, a few cm. above the apical region there is a perforation of the septum about 6x3 mm. The margins are a little irregular, somewhat rounded and the tissue is perhaps a little softer here than elsewhere. On the endocardium of the left ventricle in the region of the dilatation there is a thin, greyish-yellow, weakly adherent layer of thrombotic material. On the right ventricular side of the opening, arising from its margins, there is a rather weakly adherent, frank columnar, thrombotic mass about 2½x2x1½ cm. The mass presents a rather thick, greyish-yellow, granular outer shell with a mottled surface. It surrounds a dirty, brownish to blackish, red, softer material. Aorta: The first portion, just above the cusps, shows a small area of arteriosclerosis; elsewhere it is fairly smooth. The ascending and descending portions show a small amount of fibrous sclerosis. The abdominal portion shows a moderate amount of fibrous sclerosis with several thin, small, calcareous plates. The great branches are negative. Pulmonary artery, Veins and Venae Cavae: Negative. Liver: Negative. Gall bladder: Contains about twelve minute, blackish-green concretions. Pancreas: Shows general, well marked, fatty infiltration. The pancreatic tissue presents as small, pale, scattered islands in the ocean of fat tissue. Spleen and suprarenals: Negative. Kidneys: Negative aside from some injection of the vessels. Pelvis, Ureters and Bladder: Negative.

*Microscopic Examination.* — Pancreas: Fat present. Islands few and far between. The islands show some hyaline degeneration of their cells. Kidneys: Acute degeneration. Liver: Moderate fatty metamorphosis of the liver cells.

*Bacteriologic Report.*—Culture of heart's blood on plain agar "Good growth of streptococci."

*Anatomic Diagnosis.*—Diabetes Mellitus; slight arteriosclerosis; arteriosclerotic occlusion of the left anterior coronary artery with an area of chronic interstitial myocarditis and degeneration and perforation of the interventricular septum; mural thrombi of the ventricles; slight hypertrophy and dilatation of the heart; septicemia (streptococcus).

## SUMMARY

A case is reported presenting an interesting example of perforation of the interventricular septum of the heart in an unusual location; the heart was normal except for a limited area of sclerosis. The patient also showed slight general arteriosclerosis and diabetes mellitus.